Inventor Search

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L47 ANSWER 1 OF 7 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:325687 HCAPLUS
TITLE: Diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis
INVENTOR(S): **Aggarwal, Bharat B.**
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 43 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2005080023	A1	20050414	US 2004-925608		20040825
PRIORITY APPLN. INFO.:			US 2003-497841P	P	20030826

AB The invention provides a method of reducing or inhibiting osteoclast development induced by the receptor for activation of nuclear factor kappa B ligand (RANKL), comprising the step of contacting said osteoclast, or a precursor of the osteoclast, with a pharmacol. ED of compds. such as curcumin , guggulsterone, 1'-acetoxychavicol or analogs thereof.

IC ICM A61K031-56
INCL 514026000; 514169000
CC 1-12 (Pharmacology)
ST osteoclastogenesis inhibition diferuloylmethane guggulsterone acetoxychavicol nuclear factor kappa B; curcumin osteoclast inhibitor nuclear factor kappa B ligand
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(IκB-α (NF-κB inhibitor α); diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Phosphorylation, biological
(IκBα; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NF-κB (nuclear factor of κ light chain gene enhancer in B-cells); diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Mammary gland, neoplasm
(Paget's disease; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Bone, disease
(Paget's; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Macrophage
(RANKL-induced osteoclastogenesis; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Mammary gland, neoplasm
(adenocarcinoma; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Bone, disease
(demineralization; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)

IT Antiarthritics
Multiple myeloma
Osteoclast
Rheumatoid arthritis
(diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Carcinoma, neoplasm
(mammary adenocarcinoma; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Carcinoma, anatomical
(neck squamous cell; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Neck, anatomical
(neoplasm, squamous cell carcinoma; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Osteoporosis
(postmenopausal; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Osteoclast
(preosteoclast; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT Bone
(resorption, inhibitors; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT 159606-08-3, IκB Kinase 207621-35-0, RANKL 362516-16-3, IKKα kinase 362517-43-9, IKKβ kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT 458-37-7, Diferuloylmethane 501-92-8D, Chavicol, acetoxy derivs. 95975-55-6, Guggulsterone
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)
IT 850281-82-2
RL: PRP (Properties)
(unclaimed nucleotide sequence; diferuloylmethane, guggulsterone, and 1'-acetoxychavicol for the inhibition of osteoclastogenesis)

L47 ANSWER 2 OF 7 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:185396 HCAPLUS
DOCUMENT NUMBER: 142:254582
TITLE: Curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer
INVENTOR(S): **Aggarwal, Bharat B.**
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 31 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005049299	A1	20050303	US 2004-925814	20040825
WO 2005020908	A2	20050310	WO 2004-US27578	20040825

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,

CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2003-497842P P 20030826

AB The present invention provides a method of treating a cancerous or pre-cancerous state in an individual in need of such treatment, comprising the step of administering a pharmacol. ED of a curcuminoid to the individual. Curcumin inhibited interleukin 6-induced proliferation of human **multiple myeloma** cells.

IC ICM A61K031-12
INCL 514456000; 514689000
CC 1-6 (Pharmacology)
ST curcuminoid selective inhibitor STAT3 cancer precancer; curcumin inhibition interleukin 6 **multiple myeloma** proliferation
IT Lymphoma
(B-cell, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Lymphoma
(Burkitt's, EBV-related, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Cell nucleus
(STAT-3 translocation to, curcumin inhibition of, in **multiple myeloma** cells; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(STAT1 (signal transducer and activator of transcription 1), curcumin inhibition of IFN-α-induced phosphorylation of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Transcription factors
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)
(STAT3 (signal transducer and activator of transcription 3); curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Skin, neoplasm
(T-cell lymphoma, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Leukemia
Lymphoma
(T-cell, adult, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Leukemia
(acute lymphocytic, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Leukemia
(acute myelogenous, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Pancreas, neoplasm

(adenocarcinoma, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Lymphoma
(anaplastic, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Drug resistance
(antitumor, of **multiple myeloma** to dexamethasone, curcumin inhibition of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Ovary, neoplasm
Prostate gland, neoplasm
(carcinoma, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Leukemia
(chronic lymphocytic, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Interleukin 6
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)
(curcumin inhibition of STAT-3 phosphorylation induced by; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Antitumor agents
Combination chemotherapy
Human
Neoplasm
Signal transduction, biological
(curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Lymphoma
(cutaneous T-cell, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Ketones, biological studies
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(diketones, unsatd., curcuminoids; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Leukemia
(erythroleukemia, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Mycosis
(fungoides, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Chemotherapy
(further administration of agents for; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Carcinoma
(head squamous cell, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Carcinoma
(hepatocellular, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Liver, neoplasm
(hepatoma, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Leukemia
(large granular lymphocytic, treatment of; curcuminoids as selective

inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Skin, neoplasm
(mycosis fungoides, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Carcinoma
(neck squamous cell, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Neoplasm
(neck, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Neck, anatomical
(neoplasm, squamous cell carcinoma, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Neck, anatomical
(neoplasm, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Cell proliferation
(of **multiple myeloma** cells, STAT-3 phosphorylation linked to; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Carcinoma
(ovarian, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Carcinoma
(pancreatic adenocarcinoma, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Carcinoma
(prostatic, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Phosphorylation, biological
(protein, of STAT-3, curcumin inhibition of, in **multiple myeloma** cells; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Cell
(reduction of activated STAT3 expression in; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Kidney, neoplasm
(renal cell carcinoma, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Carcinoma
(renal cell, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Antitumor agents
(resistance to, of **multiple myeloma** to dexamethasone, curcumin inhibition of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Head, neoplasm
(squamous cell carcinoma, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Carcinoma
(squamous cell, SCCHN, treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or

precancer)
IT Brain, neoplasm
Head, neoplasm
Hodgkin's disease
Leukemia
Lung, neoplasm
Lymphoma
Mammary gland, neoplasm
Melanoma
Multiple myeloma
Polycythemia vera
(treatment of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT Interferons
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(α, curcumin inhibition of STAT-1 phosphorylation induced by; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT 50-02-2, Dexamethasone 51-21-8, 5FU 148-82-3, Melphalan 15663-27-1, Cisplatin 23214-92-8, Doxorubicin 33069-62-4, Paclitaxel 95058-81-4, Gemcitabine
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(coadministration of; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT 458-37-7, Curcumin 458-37-7D, Curcumin, analogs 22608-11-3, Demethoxycurcumin 22608-11-3D, Demethoxycurcumin, analogs 33171-05-0, Bisdemethoxycurcumin 33171-05-0D, Bisdemethoxycurcumin, analogs
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT 400628-16-2
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibition of STAT-3 phosphorylation and U266 cell growth; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)
IT 244283-56-5
RL: PRP (Properties)
(unclaimed sequence; curcuminoids as selective inhibitors of STAT-3 activation and uses in treating cancer or precancer)

L47 ANSWER 3 OF 7 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:1000246 HCAPLUS
DOCUMENT NUMBER: 142:190006
TITLE: Role of resveratrol in prevention and therapy of cancer: preclinical and clinical studies
AUTHOR(S): **Aggarwal, Bharat B.**; Bhardwaj, Anjana; Aggarwal, Rishi S.; Seeram, Navindra P.; Shishodia, Shishir; Takada, Yasunari
CORPORATE SOURCE: Cytokine Research Laboratory, Department of Bioimmunotherapy, The University of Texas M.D. Anderson Cancer Center, Houston, TX, 77030, USA
SOURCE: Anticancer Research (2004), 24(5A), 2783-2840
CODEN: ANTRD4; ISSN: 0250-7005
PUBLISHER: International Institute of Anticancer Research
DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Resveratrol, trans-3,5,4'-trihydroxystilbene, was first isolated in 1940 as a constituent of the roots of white hellebore (Veratrum grandiflorum O. Loes), but has since been found in various plants, including grapes, berries and peanuts. Besides cardioprotective effects, resveratrol exhibits anticancer properties, as suggested by its ability to suppress-proliferation of a wide variety of tumor cells, including lymphoid and myeloid cancers; **multiple myeloma** ; cancers of the: breast, prostate, stomach, colon, pancreas, and thyroid; melanoma; head and neck squamous cell carcinoma; ovarian carcinoma; and cervical carcinoma. The growth-inhibitory effects of resveratrol are mediated through cell-cycle arrest; upregulation of p21Cip/WAF1, p53 and Bax; down-regulation of survivin, cyclin D1, cyclin E, Bcl-2, Bcl-xL and cIAPs; and activation of caspases. Resveratrol has been shown to suppress the activation of several transcription factors, including NF-κB, AP-1 and Egr-1; to inhibit protein kinases including IαBa kinase, JNK, MAPK, Akt, PKC, PKD and casein kinase II,, and to down-regulate products of genes such as COX-2, 5-LOX, VEGF, IL-1, IL-6, IL-8, AR and PSA. These activities account for the suppression of angiogenesis by this stilbene. Resveratrol also has been shown to potentiate the apoptotic effects of cytokines (e.g., TRAIL), chemotherapeutic agents and γ-radiation. Pharmacokinetic studies revealed that the target organs of resveratrol are liver and kidney, where it is concentrated after absorption and is mainly converted to a sulfated form and a glucuronide conjugate. In vivo, resveratrol blocks the multistep process of carcinogenesis at various stages: it blocks carcinogen activation by inhibiting aryl hydrocarbon-induced CYP1A1 expression and activity, and suppresses tumor initiation, promotion and progression. Besides chemopreventive effects, resveratrol appears to exhibit therapeutic effects against cancer. Limited data in humans have revealed that resveratrol is pharmacol. quite safe. Currently, structural analogs of resveratrol with improved bioavailability are being pursued as potential therapeutic agents for cancer.

CC 1-0 (Pharmacology)

ST review resveratrol anticancer apoptosis cell signal transduction

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(AP-1 (activator protein 1); resveratrol inhibited tumor growth by suppressing activation of nuclear factor activator protein-1)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(BCL10; resveratrol inhibited tumor growth through cell cycle arrest by down regulation of clAPs)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Bax; resveratrol inhibited tumor growth through cell cycle arrest by up regulation of Bax)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Bcl-2; resveratrol inhibited tumor growth through cell cycle arrest by down regulation of Bcl-2)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Bcl-xL; resveratrol inhibited tumor growth through cell cycle arrest by down regulation of Bcl-xL)

IT Cyclins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(D1; resveratrol inhibited tumor growth through cell cycle arrest by down regulation of cyclin D1)

IT Cyclins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(E; resveratrol inhibited tumor growth through cell cycle arrest by down regulation of cyclin E)
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Egr-1; resveratrol inhibited tumor growth by suppressing activation of nuclear factor early growth response gene-1)
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NF-κB (nuclear factor of κ light chain gene enhancer in B-cells); resveratrol inhibited tumor growth by suppressing activation of nuclear factor kappa B)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(TRAIL (tumor necrosis factor-related apoptosis-inducing ligand); resveratrol potentiated apoptotic effect of cytokine tumor necrosis factor-related apoptosis-inducing ligand)
IT Ovary, neoplasm
(carcinoma; resveratrol showed anti proliferative effect on ovarian carcinoma through cell cycle arrest, apoptosis and suppression of transcription factor activation)
IT Uterus, neoplasm
(cervix, carcinoma; resveratrol showed anti proliferative effect on cervical carcinoma through cell cycle arrest, apoptosis and suppression of transcription factor activation)
IT Carcinoma
(cervix; resveratrol showed anti proliferative effect on cervical carcinoma through cell cycle arrest, apoptosis and suppression of transcription factor activation)
IT Intestine, neoplasm
(colon; resveratrol showed anti proliferative effect on colon cancer through cell cycle arrest, apoptosis and suppression of transcription factor activation)
IT Carcinoma
(head squamous cell; resveratrol showed anti proliferative effect on head and neck squamous cell carcinoma through cell cycle arrest, apoptosis and suppression of transcription factor activation)
IT Liver
(liver was target organ for resveratrol where it got concentrated after absorption, was converted to sulfated form, glucuronide conjugate)
IT Carcinoma
(neck squamous cell; resveratrol showed anti proliferative effect on head and neck squamous cell carcinoma through cell cycle arrest, apoptosis and suppression of transcription factor activation)
IT Neck, anatomical
(neoplasm, squamous cell carcinoma; resveratrol showed anti proliferative effect on head and neck squamous cell carcinoma through cell cycle arrest, apoptosis and suppression of transcription factor activation)
IT Carcinoma
(ovarian; resveratrol showed anti proliferative effect on ovarian carcinoma through cell cycle arrest, apoptosis and suppression of transcription factor activation)
IT Cyclin dependent kinase inhibitors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(p21CIP1; resveratrol inhibited tumor growth through cell cycle arrest by up regulation of p21Cip1/WAF1)
IT Human

(resveratrol blocked carcinogen activation by inhibiting aryl hydrocarbon-induced cytochrome P 450 1A1 expression, activity and suppressed tumor initiation, promotion, progression in human)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth by inhibiting protein kinases including IκBα kinase, JNK, MAPK, Akt, PKC, PKD and casein kinase II)

IT p53 (protein)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth through cell cycle arrest by up regulation of p 53)

IT Cytokines
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol potentiated apoptotic effect of cytokine)

IT Gamma ray
(resveratrol potentiated apoptotic effect of gamma-radiation)

IT Androgen receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol showed anti angiogenesis activity by down regulating gene product of androgen receptor)

IT Interleukin 1
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol showed anti angiogenesis activity by down regulating gene product of interleukin-1)

IT Interleukin 6
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol showed anti angiogenesis activity by down regulating gene product of interleukin-6)

IT Prostate-specific antigen
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol showed anti angiogenesis activity by down regulating gene product of prostate specific antigen)

IT Mammary gland, neoplasm
(resveratrol showed anti proliferative effect on breast cancer through cell cycle arrest, apoptosis and suppression of transcription factor activation)

IT Lymphoma
(resveratrol showed anti proliferative effect on lymphoid cancers through cell cycle arrest, apoptosis and suppression of transcription factor activation)

IT Melanoma
(resveratrol showed anti proliferative effect on melanoma through cell cycle arrest, apoptosis and suppression of transcription factor activation)

IT **Multiple myeloma**
(resveratrol showed anti proliferative effect on **multiple myeloma** through cell cycle arrest, apoptosis and suppression of transcription factor activation)

IT Pancreas, neoplasm
(resveratrol showed anti proliferative effect on pancreas cancer through cell cycle arrest, apoptosis and suppression of transcription factor activation)

IT Prostate gland, neoplasm
(resveratrol showed anti proliferative effect on prostate cancer through cell cycle arrest, apoptosis and suppression of transcription factor activation)

IT Stomach, neoplasm
(resveratrol showed anti proliferative effect on stomach cancer through

cell cycle arrest, apoptosis and suppression of transcription factor activation)

IT Thyroid gland, neoplasm
(resveratrol showed anti proliferative effect on thyroid cancer through cell cycle arrest, apoptosis and suppression of transcription factor activation)

IT Apoptosis
(resveratrol showed anti proliferative effect through induction of apoptosis in cell lines of various origin namely leukemia, breast, prostate, colon, pancreas, ovary, thyroid, cervical, head and neck squamous cell)

IT Cell cycle
(resveratrol showed anti proliferative effect through induction of cell cycle arrest in cell lines of various origin namely leukemia, breast, prostate, colon, pancreas, ovary, thyroid, cervical, head and neck squamous cell)

IT Antitumor agents
Neoplasm
(resveratrol showed chemopreventive activity and therapeutic effect against cancer by its ability to suppress proliferation mediated via cell cycle arrest, apoptosis and suppression of transcription factor activation)

IT Interleukin 8
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol sowed anti angiogenesis activity by down regulating gene product of interleukin-8)

IT Head, neoplasm
(squamous cell carcinoma; resveratrol showed anti proliferative effect on head and neck squamous cell carcinoma through cell cycle arrest, apoptosis and suppression of transcription factor activation)

IT Signal transduction, biological
(structural analogs of resveratrol with improved bioavailability may be potential therapeutic agents for cancer)

IT 329764-85-4, Cytochrome P 450 1A1
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol blocked carcinogen activation by inhibiting aryl hydrocarbon-induced cytochrome P 450 1A1 expression, activity and suppressed tumor initiation, promotion, progression in human)

IT 329900-75-6, Cyclo oxygenase-2
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth by down regulating gene product of cyclo oxygenase-2)

IT 366806-33-9, Casein kinase II
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth by inhibiting casein kinase II)

IT 148640-14-6, Akt protein kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth by inhibiting protein kinase Akt)

IT 141436-78-4, Protein kinase C
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth by inhibiting protein kinase C)

IT 161384-20-9, Protein kinase D
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth by inhibiting protein kinase D)

IT 362516-16-3, IκBα Kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth by inhibiting protein kinase IκBα kinase)

IT 155215-87-5, JNK

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth by inhibiting protein kinase JNK)
IT 142243-02-5
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth by inhibiting protein kinase mitogen-activated protein kinase)
IT 186322-81-6, Caspase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth through cell cycle arrest by activation of caspases)
IT 371761-91-0, Survivin
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol inhibited tumor growth through cell cycle arrest by down regulation of survivin)
IT 127464-60-2, Vascular endothelial growth factor
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resveratrol showed anti angiogenesis activity by down regulating gene product of vascular endothelial growth factor)
IT 501-36-0, Resveratrol
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); PKT (Pharmacokinetics); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(resveratrol showed chemopreventive activity and therapeutic effect against cancer by its ability to suppress proliferation mediated via cell cycle arrest, apoptosis and suppression of transcription factor activation)
REFERENCE COUNT: 370 THERE ARE 370 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 4 OF 7 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:701795 HCAPLUS
DOCUMENT NUMBER: 141:200229
TITLE: Inhibitors of receptor activator of NF-kappaB (RANK) and uses thereof
INVENTOR(S): **Aggarwal, Bharat B.**; Darnay, Bryant G.; Singh, Sujay
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 33 pp., Cont.-in-part of U.S. Ser. No. 143,293.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2004167072	A1	20040826	US 2004-786316		20040225
US 2003013170	A1	20030116	US 2002-143293		20020510
PRIORITY APPLN. INFO.:			US 2001-290429P	P	20010511
			US 2002-143293	A2	20020510

AB The invention provides a RANK (receptor activator of NF-κB) inhibitor consisting of a TRAF-6 (TNF receptor-associated factor-6) binding domain attached to a leader sequence. The decoy peptide inhibits RANKL (RANK ligand)-mediated osteoclast differentiation, thus indicating that targeted disruption of interaction between RANK and TRAF6 may prove useful as a therapeutic for metabolic bone disorders, leukemia, arthritis, and metastatic cancer of the bone.

IC ICM A61K038-16
ICS C07K014-16
INCL 514012000; 530350000
CC 1-12 (Pharmacology)
ST RANK inhibitor TRAF6 binding domain therapeutic; bone disease arthritis treatment RANK inhibitor; leukemia bone cancer treatment RANK inhibitor
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Antennapedia, Drosophila antennapedia signal peptide sequence; RANK inhibitors and therapeutic uses)
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GCN4, signal sequence; RANK inhibitors and therapeutic uses)
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NF-κB (nuclear factor of κ light chain gene enhancer in B-cells); RANK inhibitors and therapeutic uses)
IT Cytokine receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(RANK (receptor activator of NF-κB); RANK inhibitors and therapeutic uses)
IT Antiarthritics
Antitumor agents
Arthritis
Drug delivery systems
Drug screening
Genetic vectors
Human
Human T-lymphotropic virus 2
Leukemia
Monocyte
Multiple myeloma
Signal transduction, biological
Viral vectors
Yeast
(RANK inhibitors and therapeutic uses)
IT CD40 (antigen)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(RANK inhibitors and therapeutic uses)
IT Peptides, biological studies
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(RANK inhibitors and therapeutic uses)
IT Leader peptides
Signal peptides
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(RANK inhibitors and therapeutic uses)
IT Molecular association
(RANK-TRAF6; RANK inhibitors and therapeutic uses)
IT Ligands
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(RANKL; RANK inhibitors and therapeutic uses)
IT Animal cell line
(RAW264.7; RANK inhibitors and therapeutic uses)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(RIP2; RANK inhibitors and therapeutic uses)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(TRAF6 (tumor necrosis factor receptor-associated factor 6); RANK inhibitors and therapeutic uses)
IT DNA
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(binding peptide, signal sequence; RANK inhibitors and therapeutic uses)
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(c-fos, signal sequence; RANK inhibitors and therapeutic uses)
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(c-jun, signal sequence; RANK inhibitors and therapeutic uses)
IT Flock house virus
(coat protein, signal sequence; RANK inhibitors and therapeutic uses)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(coat, signal sequence; RANK inhibitors and therapeutic uses)
IT Osteoclast
(differentiation, inhibition; RANK inhibitors and therapeutic uses)
IT Brome mosaic virus
(gag; RANK inhibitors and therapeutic uses)
IT Drug delivery systems
(liposomes; RANK inhibitors and therapeutic uses)
IT Bone, neoplasm
(metastasis; RANK inhibitors and therapeutic uses)
IT Crystal structure
(of TRAF6-binding peptide complex; RANK inhibitors and therapeutic uses)
IT Mammary gland, neoplasm
(osteoclast differentiation induced by; RANK inhibitors and therapeutic uses)
IT Cell differentiation
(osteoclast, inhibition; RANK inhibitors and therapeutic uses)
IT Bone, disease
(osteolysis; RANK inhibitors and therapeutic uses)
IT Neoplasm
Prostate gland, neoplasm
(osteolytic lesion induced by; RANK inhibitors and therapeutic uses)
IT Bone, disease
(osteopenia; RANK inhibitors and therapeutic uses)
IT Conformation
(protein; RANK inhibitors and therapeutic uses)
IT Rev protein
gag proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(signal sequence; RANK inhibitors and therapeutic uses)
IT Human immunodeficiency virus 1
(tat and rev; RANK inhibitors and therapeutic uses)
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(tat, signal sequence; RANK inhibitors and therapeutic uses)
IT Virus
(viral RNA binding peptide, signal sequence; RANK inhibitors and therapeutic uses)
IT RNA
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(viral RNA binding peptide, signal sequence; RANK inhibitors and therapeutic uses)
IT Peptides, biological studies

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(viral RNA binding, signal sequence; RANK inhibitors and therapeutic uses)
IT Amino acids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(D-; RANK inhibitors and therapeutic uses)
IT 62031-54-3, Fibroblast growth factor
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Kaposi, signal sequence; RANK inhibitors and therapeutic uses)
IT 74-79-3, L-Arginine, biological studies 137632-07-6, p44 Erk kinase 137632-08-7, p42 Erk kinase 165245-96-5, p38 Map kinase 167397-96-8, IRAK-1 kinase 200578-48-9, IRAK-2 kinase 241825-09-2, IRAK-M kinase 289898-51-7, Jnk1 kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(RANK inhibitors and therapeutic uses)
IT 475556-80-0 475556-81-1
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(RANK inhibitors and therapeutic uses)
IT 447459-03-2 475556-63-9 475556-64-0 475556-65-1 475556-66-2
475556-67-3 475556-68-4 475556-69-5 475556-70-8 475556-71-9
475556-72-0 475556-73-1 475556-74-2 475556-75-3 475556-76-4
475556-77-5 475556-78-6 475556-79-7 743919-32-6
RL: PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(RANK inhibitors and therapeutic uses)
IT 96337-25-6 148796-86-5 165893-48-1
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(RANK inhibitors and therapeutic uses)

L47 ANSWER 5 OF 7 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:342327 HCAPLUS
DOCUMENT NUMBER: 140:368302
TITLE: Nuclear factor-κB and STAT3 are constitutively active in CD138+ cells derived from **multiple myeloma** patients, and suppression of these transcription factors leads to apoptosis
AUTHOR(S): Bharti, Alok C.; Shishodia, Shishir; Reuben, James M.; Weber, Donna; Alexanian, Raymond; Raj-Vadhan, Saroj; Estrov, Zeev; Talpaz, Moshe; **Aggarwal, Bharat B.**
CORPORATE SOURCE: Departments of Bioimmunotherapy, Hematopathology, The University of Texas M. D. Anderson Cancer Center, Houston, TX, 77030, USA
SOURCE: Blood (2004), 103(8), 3175-3184
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: American Society of Hematology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Chemoresistance is a major problem in the treatment of patients with **multiple myeloma** (MM). Because of the central role of the nuclear transcription factors nuclear factor-κB (NF-κB) and signal transducer and activator of transcription 3 (STAT3) in chemoresistance, cell survival, and proliferation, we investigated whether MM cells derived from patients express activated NF-κB and STAT3 and if their suppression induces apoptosis. We assayed CD138+ cells from the bone marrow of 22 MM patients and checked for the activated forms of NF-κB and STAT3 by immunocytochem. We found that MM cells from all the patients expressed the activated forms of NF-κB and STAT3 but to

a variable degree (NF-κB: low, 3 of 22; moderate, 5 of 22; or high, 14 of 22; STAT3: none, 1 of 22; low, 3 of 22; moderate, 5 of 22; or high, 14 of 22). Constitutive activation of NF-κB was in some cases also independently confirmed by electrophoretic mobility gel shift assay. In contrast to MM patients, activated forms of NF-κB and STAT3 were absent in cells from healthy individuals. Suppression of NF-κB and STAT3 activation in MM cells by ex vivo treatment with curcumin (diferuloylmethane) resulted in a decrease in adhesion to bone marrow stromal cells, cytokine secretion, and in the viability of cells. When compared with curcumin, dexamethasone was less effective in suppression of NF-κB activation and induction of apoptosis in myeloma cells. Overall, our results indicate that fresh cells from MM patients express constitutively active NF-κB and STAT3, and suppression of these transcription factors inhibits the survival of the cells.

CC 1-6 (Pharmacology)
Section cross-reference(s): 14

ST myeloma NFkappaB STAT3 inhibitor apoptosis

IT Antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(CD138, pos. cells; myeloma-derived NF-κB and STAT3 suppression leads to apoptosis)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NF-κB (nuclear factor of κ light chain gene enhancer in B-cells); myeloma-derived NF-κB and STAT3 suppression leads to apoptosis)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(STAT3 (signal transducer and activator of transcription 3); myeloma-derived NF-κB and STAT3 suppression leads to apoptosis)

IT Antitumor agents
Apoptosis
Drug resistance
Drug targets
Human
Multiple myeloma
(myeloma-derived NF-κB and STAT3 suppression leads to apoptosis)

IT 50-02-2, Dexamethasone 458-37-7, Curcumin
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(myeloma-derived NF-κB and STAT3 suppression leads to apoptosis)

REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 6 OF 7 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:790896 HCAPLUS
DOCUMENT NUMBER: 139:390906
TITLE: Curcumin (diferuloylmethane) inhibits constitutive and IL-6-inducible STAT3 phosphorylation in human **multiple myeloma** cells
AUTHOR(S): Bharti, Alok C.; Donato, Nicholas; **Aggarwal, Bharat B.**
CORPORATE SOURCE: Cytokine Research Section, Department of Bioimmunotherapy, Unit 143, University of Texas M. D. Anderson Cancer Center, Houston, TX, 77030, USA
SOURCE: Journal of Immunology (2003), 171(7), 3863-3871
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal

LANGUAGE: English

AB Numerous reports suggest that IL-6 promotes survival and proliferation of **multiple myeloma** (MM) cells through the phosphorylation of a cell signaling protein, STAT3. Thus, agents that suppress STAT3 phosphorylation have potential for the treatment of MM. In the present report, we demonstrate that curcumin (diferuloylmethane), a pharmacol. safe agent in humans, inhibited IL-6-induced STAT3 phosphorylation and consequent STAT3 nuclear translocation. Curcumin had no effect on STAT5 phosphorylation, but inhibited the IFN-α-induced STAT1 phosphorylation. The constitutive phosphorylation of STAT3 found in certain MM cells was also abrogated by treatment with curcumin. Curcumin-induced inhibition of STAT3 phosphorylation was reversible. Compared with AG490, a well-characterized Janus kinase 2 inhibitor, curcumin was a more rapid (30 min vs 8 h) and more potent (10 μM vs 100 μM) inhibitor of STAT3 phosphorylation. In a similar manner, the dose of curcumin completely suppressed proliferation of MM cells; the same dose of AG490 had no effect. In contrast, a cell-permeable STAT3 inhibitor peptide that can inhibit the STAT3 phosphorylation mediated by Src blocked the constitutive phosphorylation of STAT3 and also suppressed the growth of myeloma cells. TNF-α and lymphotoxin also induced the proliferation of MM cells, but through a mechanism independent of STAT3 phosphorylation. In addition, dexamethasone-resistant MM cells were found to be sensitive to curcumin. Overall, our results demonstrated that curcumin was a potent inhibitor of STAT3 phosphorylation, and this plays a role in the suppression of MM proliferation.

CC 1-6 (Pharmacology)

ST curcumin STAT3 phosphorylation myeloma antitumor interleukin 6 signaling

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(STAT1 (signal transducer and activator of transcription 1); curcumin inhibits STAT3 phosphorylation in human **multiple myeloma** cells)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(STAT3 (signal transducer and activator of transcription 3); curcumin inhibits STAT3 phosphorylation in human **multiple myeloma** cells)

IT Proteins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(STAT3 inhibitor peptide; STAT3iP; curcumin inhibits STAT3 phosphorylation in human **multiple myeloma** cells)

IT Antitumor agents
Human
Multiple myeloma
Phosphorylation, biological
(curcumin inhibits STAT3 phosphorylation in human **multiple myeloma** cells)

IT Interleukin 6
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(curcumin inhibits STAT3 phosphorylation in human **multiple myeloma** cells)

IT Interferons
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(α; curcumin inhibits STAT3 phosphorylation in human **multiple myeloma** cells)

IT 458-37-7, Curcumin 133550-30-8, AG490
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(curcumin inhibits STAT3 phosphorylation in human **multiple myeloma** cells)
REFERENCE COUNT: 73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 7 OF 7 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:90410 HCAPLUS
DOCUMENT NUMBER: 139:30341
TITLE: Curcumin (diferuloylmethane) down-regulates the constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis
AUTHOR(S): Bharti, Alok C.; Donato, Nicholas; Singh, Sujay; **Aggarwal, Bharat B.**
CORPORATE SOURCE: Cytokine Research Section, Department of Bioimmunotherapy, The University of Texas MD Anderson Cancer Center, Houston, TX, 77030, USA
SOURCE: Blood (2003), 101(3), 1053-1062
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: American Society of Hematology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Because of the central role of the transcription factor nuclear factor-κB (NF-κB) in cell survival and proliferation in human **multiple myeloma** (MM), we explored the possibility of using it as a target for MM treatment by using curcumin (diferuloylmethane), an agent known to have very little or no toxicity in humans. We found that NF-κB was constitutively active in all human MM cell lines examined and that curcumin, a chemopreventive agent, down-regulated NF-κB in all cell lines as indicated by electrophoretic mobility gel shift assay and prevented the nuclear retention of p65 as shown by immunocytochem. All MM cell lines showed constitutively active IκB kinase (IKK) and IκBα phosphorylation. Curcumin suppressed the constitutive IκBα phosphorylation through the inhibition of IKK activity. Curcumin also down-regulated the expression of NF-κB-regulated gene products, including IκBα, Bcl-2, Bcl-xL, cyclin D1, and interleukin-6. This led to the suppression of proliferation and arrest of cells at the G1/S phase of the cell cycle. Suppression of NF-κB complex by IKKγ/NF-κB essential modulator-binding domain peptide also suppressed the proliferation of MM cells. Curcumin also activated caspase-7 and caspase-9 and induced polyadenosine-5'-diphosphate-ribose polymerase (PARP) cleavage. Curcumin-induced down-regulation of NF-κB, a factor that has been implicated in chemoresistance, also induced chemosensitivity to vincristine and melphalan. Overall, our results indicate that curcumin down-regulates NF-κB in human MM cells, leading to the suppression of proliferation and induction of apoptosis, thus providing the mol. basis for the treatment of MM patients with this pharmacol. safe agent.
CC 1-6 (Pharmacology)
ST curcumin NFkappaB kinase myeloma apoptosis gene
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Bcl-2; curcumin (diferuloylmethane) down-regulates constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis)
IT Proteins

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Bcl-xL; curcumin (diferuloylmethane) down-regulates constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis)

IT Cyclins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(D1; curcumin (diferuloylmethane) down-regulates constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(IκB-α (NF-κB inhibitor α); curcumin (diferuloylmethane) down-regulates constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NF-κB (nuclear factor of κ light chain gene enhancer in B-cells); curcumin (diferuloylmethane) down-regulates constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis)

IT Antitumor agents
Apoptosis
Cell cycle
Human
Multiple myeloma
(curcumin (diferuloylmethane) down-regulates constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis)

IT Gene, animal
Interleukin 6
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(curcumin (diferuloylmethane) down-regulates constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis)

IT Phosphorylation, biological
(protein; curcumin (diferuloylmethane) down-regulates constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis)

IT Drug interactions
(synergistic; curcumin (diferuloylmethane) down-regulates constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis)

IT 9055-67-8, Poly ADP-ribose polymerase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(curcumin (diferuloylmethane) down-regulates constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis)

IT 57-22-7, Vincristine 148-82-3, Melphalan 458-37-7, Curcumin
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL

(Biological study); USES (Uses)
(curcumin (diferuloylmethane) down-regulates constitutive activation of nuclear factor-κB and IκBα kinase in human **multiple myeloma** cells, leading to suppression of proliferation and induction of apoptosis)

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT